117. Smoked/salted/cured/pickled food

Only one study could be found which attempted to relate lung cancer risk to frequent consumption of foods which were smoked, salted, cured or pickled, and details of it are given in Table 117. Three relative risks, ranging from 0.92-1.60, were estimated for various food items.

It can be seen from the evidence presented in the table that there is no convincing relationship between lung cancer risk and frequent consumption of smoked, salted, cured or pickled foods.

Table 117: Estimates of relative risk for frequent consumption of smoked/salted/cured/pickled food

Study	Population	Food item	Relative risk (95% limits
Koo (1988)	Hong Kong women	Dried/salted fish	0.95
		Smoked/cured meat/poultry	0.92
		Pickled vegetables	1.60

References

1. Koo LC (1988) Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoked. Nutr Cancer, 11, 155-172.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

118. Smoky coal

Details of the only study which presented data relevant to an investigation of the possible association between lung cancer risk and use of smoky coal are given in Table 118. Two relative risks were estimated, 4.0 for men and 3.7 for women. In addition, evidence of an association between smoky coal use and lung cancer mortality rate per 100,000 population was presented, with rates ranging from 174.21 in an area where 100.0% of households burned smoky coal, down to 3.81 and 2.08 in two areas where no households did.

The data presented in the table provide quite strong evidence of an association between lung cancer risk and use of smoky coal, and it was suggested by the authors that in areas where smoking prevalence is low the burning of smoky coal may be the prime determinant of lung cancer [1]. However, it should be borne in mind that the data has been taken from one study only, and results from further studies are needed before definite conclusions can be reached.

Table 118: Estimates of relative risk/mortality rate for smoky coal

Study	Population	% Households burning smoky coal	Relative risk (95% limits)
He et al (1990)	Chinese men	-	4.0
	Women	-	3.7
	Chinese residents	100.0	174.21[1]
		89.7	128.31[1]
		81.9	104.09[1]
		78.0	22.96[1]
		76.1	39.46[1]
		49.7	13.48[1]
		35.2	7.49[1]
		34.0	19.03[1]
		2.7	9.55[1]
		0.0	3.81[1]
		0.0	2.08[1]

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

119. Social class

Table 119 gives details of the available data relevant to an investigation of the possible association between lung cancer risk and social class, according to the index of social class used.

For education, both of the studies using this index of social class found evidence of an increasing risk of lung cancer with decreasing educational level.

Respondents were classified according to their income in eight studies, with four finding an inverse relationship with lung cancer risk and another reporting a very weak relationship in this direction. Two studies actually observed a decrease in risk in lower income groups, although both of these studies divided the respondents into two groups only, while the remaining study failed to find any relationship between income and lung cancer risk.

None of the six studies which used occupational group as an index of social class found unequivocal evidence of an inverse relationship with lung cancer risk, although four did show that subjects in some of the "lower" occupational groups had an increased risk of lung cancer compared to those in "higher" groups.

Four studies grouped subjects according to the average rent in their area of residence, but although all found some evidence of an increasing risk of lung cancer with decreasing rent paid, in none was it a smooth relationship.

Of the 21 studies which classified respondents by their social class, as used in censuses and similar surveys, only two found a convincing inverse relationship with lung cancer risk. However, nine other studies presented some evidence that those in lower social classes have a higher risk of lung cancer than those in higher social classes.

Finally, socio-economic status was used to group respondents by seven studies, of which three found an inverse relationship with lung cancer risk, and two others found at least some evidence of such a relationship.

It can be seen from the table that a variety of indices were used to measure the respondents' social class, with some being more satisfactory than others. While occupational group and educational level are reasonably reliable, as they are based on data relating to the actual

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Footnote to Table 118

1 Mortality rate per 100,000 population

References

1. He X, Chapman RS, Yang R et al (1990) Lung cancer and indoor air pollution in Xuan Wei, China: Current progress. In: Kasuga H (ed.) Indoor air quality, 435-441. Springer-Verlag.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

subject, socio-economic level, income and rent may be less so. This is because for these indices classification was often based on the average for the area of residence, rather than on that pertaining to the subject specifically. Thus, such measures could be somewhat inaccurate if the subject was not average in these respects.

Although there does appear to be some evidence that those in higher social classes are at less risk of lung cancer than those of lower social class, such a relationship is far from proved.

Table 119: Estimates of relative risk/standardized mortality ratio for low social class

Population	Standardized mortality ratios			
Education level:				
	College	High school	Elementary school	<8 years education
US white men, 1960	61	95	114	118
US white women, 1960	90	94	96	123

Income level:					
	High				Low
US men, 1959-67	79	93	107	84	104[1]
US white men, 1947	67	78	99	118	134[1]
US white women, 1947	98	88	98	99	126[1]
US non-white men, 1947	-	-	45	114	127[1]
US white men, 35-54, 1959-61	23.0	25.2	36.8	48.0	60.8[2]
US white men, 55+ 1959-61	87.0	178.7	202.0	255.0	331.7[2]
US white women, 35-54 1959-61		6.5			4.5[2]
US white women, 55+ 1959-61		29.9			18.1[2]

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 119 continued

Occupation groups:

	Professional	Managerial	Clerical	Sales	Craftsmen	Operatives	Services	Labs. exc. farm	Farmers
US men, 1950	83	94	95	103	130	107	123	127	54

	Outdoor workers		Indoor workers		
	Agriculture	Constructive	Industry	Transport	Office, etc
German men, 1958-63	58	56	176	192	135[2]

	Independent		Employees				SW	UW	Unoccupied	
	Rural	Urban	I	II	III	IV			Inc HW	Exc HW
Danish men, 1970-5	44	107	51	68	107	116	135	113	181	-
Danish women 1970-5	-	107	-	-	101	102	-	115	115	99

	Professional	Managerial	Sales	Clerical	Craftsmen	Operatives	Laborers	Services
US white men, 1972-5	83	113	91	101	111	102	113	117[1]
US white women, 1972-5	103	213	73	104	92	62	177	95[1]

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 119 continued

Rent level:

	High				Low
Danish men, 1943-7	66	99	90	97	116[1]
Danish women, 1943-7	50	90	79	100	131[1]
US white men, 1948-52	35.8	31.1	44.8		57.9[3]
US white women, 1948-52	5.0	6.3	5.0		7.4[3]

Social class:

	I	II	III	IV	V	Farmers	Farm workers
US men, 1949-51	85	77	112	108	112	94	54

	I	II	III	IV	V
UK men, 1911	94	106	106	83	111
UK men, 1921	100	109	97	79	124
UK men, 1931	107	96	101	91	112
UK married women[4], 1931	100	100	110	82	91
Scottish men, 1949-53	104	81	115	86	109[5]
UK men, 1951	81	82	107	91	118
UK married women[4], 1951	119	95	102	98	96
UK single women, 1951	75	101	112	100	91
Scottish men, 1959-63	61	70	104	98	151[5]
Scottish married women[4], 1959-63	59	81	78	85	138[5]
Scottish single women, 1959-63	130	73	110	174	149[5]
UK men, 1961	53	72	107	104	148
UK married women[4], 1961	83	89	102	101	131
UK single women, 1961	108	118	110	106	127
Finnish men, 1969-72	53	89	124	153	82[6]
Finnish married women[7], 1969-72	88	105	123	123	60[6]

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 119 continued

Norwegians, 1970-73	81	113	74	122	40
UK men, 1971	53	68	110	123	143
UK married women[4], 1971	73	82	112	125	134
UK single women, 1971	82	92	123	129	114

Socio-economic level:

	High		Low
US men, 1935-49	88	84	116[1]
US women, 1935-49	82	66	139[1]
US white men, 1949-51	35.1	43.2	47.3[8]
US white women, 1949-51	5.6	6.6	7.3[8]
Australian men, 1971	41		52[8]
Australian women, 1971	9		8[8]
Turkish men, 1979-84*	1.0	1.0(0.7-1.4)	0.9(0.7-1.3)[9]

HW = housewives; SW = skilled workers; UW = unskilled workers

* From Dosemeci et al (1993); all other studies from Logan (1982)

1 Standardized incidence ratio

2 Death rate per 100,000

3 Standardized incidence rate per 100,000

4 According to husband's occupation

5 Tumours of respiratory system

6 Comparative mortality factor

7 According to own or husband's occupation

8 Standardized death rate per 100,000

9 Relative risk

References

1. Dosemeci M, Hayes RB, Vetter R (1993) Occupational physical activity, socioeconomic status, and risks of 15 cancer sites in Turkey. Cancer Causes and Control, 4, 313-321.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

120. Soots/chimney sweeps

Although exposure to soots may occur amongst heating-unit service personnel, brick masons and helpers, building demolition personnel, insulators and firemen, chimney sweeps are the principal workers receiving occupational exposure to soots. It was thus decided to combine the reports for soots and chimney sweeps to avoid unnecessary repetition. The studies which investigated the potential risks of soot exposure and employment as a chimney sweep and lung cancer are detailed in Table 120. Thirteen standardized mortality ratios were given, lying in the range 86-313, of which 11 were raised. Additionally, two proportional registration ratios, of 128 and 134, were also given.

Little information was available from the studies on the exposures of the subjects, although one study did attempt to classify cumulative exposure to soots from the number of years spent working as a chimney sweep [1]. Soot is a complex mixture produced by the combustion of coal, coke, oils, wood, paper, rubber, plastics, and resins, and has been shown to contain potentially carcinogenic metals such as arsenic, chromium, cadmium and nickel, as well as sulphur dioxide, carbon monoxide, asbestos and organic solvents [1,2]. Additionally, the character of the sweeping duties may vary considerably and this will affect the exposure of the subject. For example, an hour's climb in chimney ducts may give an exposure to particulate dust 100 to 1000 times higher than that in other duties. Therefore, it will be very difficult to estimate the exposures of the study subjects, or the effects of any one component of soot.

Even so, IARC still felt that there was "sufficient" evidence for the carcinogenicity of soots to humans [2], although no attempt was made to identify particular causative agents from among the constituent substances in soots. It would appear that employment as a chimney sweep should also be viewed as an occupation which carries a potential carcinogenic risk.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 120: Estimates of standardized mortality ratio for exposure to soot/employment as a chimney sweep

Study	Location	Subset	Standardized mortality ratio
Kennaway and Kennaway (1936, 1947)[1]	E/W	1921-1932	169($p<0.05$)[2]
		1933-1938	90($p<0.05$)[2]
Kupetz (1966)[1]	GDR		>100
OPCS (1978)	E/W	Men aged 15-64[3], 1970-2	109
		Aged 65-74, 1970-2	116
		Aged 15-74, 1966-7	134($p<0.01$)[4]
		Aged 15-74, 1968-9	128($p<0.01$)[4]
Hogstedt et al (1982)[1]	Sweden		232($p<0.05$)[2]
Logan (1982)	UK	Men, 1951	164
		1961	123
		1971	109
		Married women[5] - 1971	86
Hansen (1983)[1]	Denmark		313($p<0.05$)[2]
Hogstedt et al (1983)[1]	Sweden		277($p<0.05$)[2]
Evanoff et al (1993)	Sweden		206(154-269)

E/W = England/Wales; GDR = German Democratic Republic

1 From International Agency for Research on Cancer (1985)

2 Estimated from data given

3 Includes charwomen, office cleaners, window cleaners, chimney sweeps

4 Proportional registration ratio

5 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

References

1. Evanoff BA, Gustavsson P and Hogstedt C (1993) Mortality and incidence of cancer in a cohort of Swedish chimney sweeps: An extended follow up study. Br J Ind Med, 50, 450-459.

2. International Agency for Research on Cancer (1985) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 35: Polynuclear aromatic compounds, part 4, bitumens, coal-tars and derived products, shale-oils and soots, 219-241. IARC, Lyon.

3. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London.

4. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

121. Spices

Only one study could be found which attempted to relate the risk of lung cancer to consumption of spices and it is detailed in Table 121. It was reported that bronchial cancer cases used less spices than controls.

With so little data, it is not possible to determine whether a protective effect of spices exists regarding lung cancer risk.

Table 121: Observations for spice consumption

Study	Population	Observations
Denoix et al (1958)	French men	Bronchial cancer cases consumed less spices than controls

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

122. Sugar

Details of the only study found which investigated the possible relationship between lung cancer risk and sugar consumption are given in Table 122. It was observed that cancer cases consumed more sugar than controls.

In the absence of other supporting evidence it is not possible to evaluate the potential carcinogenicity of sugar to humans.

Table 122: Observations on sugar consumption

Study	Population	Observations
Denoix et al (1958)	French men	Bronchial cancer cases consumed more sugar than controls

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

123. Sugarcane farmers

Table 123 gives details of the only study found which contained data relevant to the investigation of lung cancer risk among sugarcane farmers. A relative risk of 2.4 was estimated.

The occurrence of cases of mesothelioma among the study population led to the suggestion that exposure to fibrous cane dust may be responsible for the increased rates of lung disease observed [1], but no objective measures of the level of such dust were made by the study. Results from more studies will be needed before the carcinogenicity of exposures associated with sugarcane farming can be evaluated.

Table 123: Estimate of relative risk for sugarcane farmers

Study	Location	Relative risk (95% limits)
Rothschild and Mulvey (1982)[1]	USA	2.4

1 From Williams Pickle (1984)

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. Williams Pickle L, Correa P and Fontham E (1984) Recent case-control studies of lung cancer in the United States. In: Lung cancer: Causes and prevention, 101-115. Verlag Chemie International Inc.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

124. Taking saunas

Table 124 gives details of the only study found which attempted to relate lung cancer risk to sauna usage. A relative risk of 1.7 was found for men who were heavy smokers.

It was suggested that the increase in lung cancer risk observed may be due to the production of polycyclic aromatic hydrocarbons during the wood heating, and to asbestos, used as insulation, in the ambient air. However, in the absence of any other data, it is not really possible to evaluate the potential carcinogenicity of saunas.

Table 124: Estimate of relative risk for taking saunas

Study	Population	Relative risk
Tenkanen et al (1985)	Finnish male heavy smokers	1.7

References

1. Tenkanen L, Hakulinen T, Hakama M and Saxen E (1985) Sauna, dust and migration as risk factors in lung cancer among smoking and non-smoking males in Finland. Int J Cancer, 35, 637-642.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

125. Talc

Table 125 details the seven studies which attempted to relate lung cancer risk to exposure to talc. Five standardized mortality ratios were calculated, and these ranged from 46-290, with all but one being raised. Two relative risks, of 1.00 and 3.24, were also estimated.

Only two studies (Kleinfeld, Selevan) provided objective data on levels of exposure to talc, although these were estimated in the study by Selevan. It is possible that inaccuracies due to misclassification of exposure could have occurred in the other studies. In addition, talc deposits contain various other minerals, including carbonates, free silica, serpentines (including chrysotile) and amphibole minerals (asbestiform and non-asbestiform), which may be potential carcinogens themselves. All of the studies appeared to have considered this to some extent, and gave information on the content of other minerals, although three studies noted that there was no exposure to asbestos (Leophonte, Rubino, Selevan). However, as most of the exposures to other minerals occurred in conjunction with exposure to talc, it was not possible to analyze the effects of talc alone. Therefore, the observed associations may have been brought about by exposure to one of these other minerals.

When evaluating the carcinogenicity of talc to humans, IARC felt that the evidence for talc not containing asbestiform fibres was "inadequate", while that for talc containing asbestiform fibres was "adequate" [2,3]. Meanwhile, a review of lung cancer and occupational exposures described talc as only a "possible" carcinogen, although no attempt was made to classify talc containing and not containing asbestiform fibres separately [1].

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

126. Tea drinking

Table 126 gives details of the six studies which attempted to relate the risk of lung cancer to tea consumption. Seven relative risks were estimated, ranging from 0.84-2.74, of which five were above 1.00. In addition, two studies reported finding a positive correlation between lung cancer mortality, or incidence, and tea consumption.

Over 400 volatile compounds have been identified in black teas, compared to some 200 in green teas, but with the exception of the study by Tewes et al, no attempt was made to distinguish between the different types. Furthermore, information on the consumption of other hot beverages was not given by any of the studies. Therefore, non-drinkers of tea could be drinkers of other hot beverages, such as coffee.

In the conclusions of their study, Tewes et al described tea as a "low risk agent" for lung cancer in humans [3], while IARC felt there was "inadequate evidence" for the carcinogenicity to humans of tea drinking [2].

Table 126: Estimates of relative risk for tea drinking

Study	Population	Relative risk (95% limits)
Stocks (1970)	Women in 20 countries	Positive correlation between lung cancer mortality and tea consumption
Hirayama (1974)	Japanese men	0.84
Armstrong and Doll (1975)[1]	Women in 23 countries	Positive correlation between lung cancer incidence and tea consumption
Dean et al (1977)[2]	UK men	1.22($p<0.01$)
	Women	1.65($p<0.01$)
Kinlen et al (1988)	UK men	1.4

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 125: Estimates of standardized mortality ratio/relative risk for exposure to talc

Study	Population	Standardized mortality ratio
Kleinfeld et al (1974)[1]	US talc miners/millers	3.24*[2]
Rubino et al (1976)[1]	Italian talc miners/millers	46[2]
Brown et al (1979)[1]/Dement et al (1980)[1]	US talc miners/millers	290($p<0.05$)[3]
Selevan et al (1979)[1]	US talc miners/millers	163($p>0.05$)[2]
Stille and Tabershaw (1982)[1]	US talc miners/millers	157
Leophonte et al (1983)[1]	French talc workers	1.00*
Thomas and Stewart (1987)[4]	Pottery workers	143

* Relative risk

1 From International Agency for Reseach on Cancer (1987a)

2 Estimated from data given

3 Tumours of respiratory system

4 From International Agency for Reseach on Cancer (1987b)

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. International Agency for Research on Cancer (1987a) Monographs on the evaluation of carcinogenic risks to humans. Volume 42: Silica and some silicates, 185-224. IARC, Lyon.

3. International Agency for Research on Cancer (1987b) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 349-350. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 126 continued

Tewes et al (1990)	Hong Kong women - black tea	1.43(0.88-2.33)
	Green tea	2.74(1.10-6.80)
Oguni et al (1992)[3]	Japanese residents	<1.00

1 From International Agency for Research on Cancer (1991)

2 From Tewes et al (1990)

3 From Yang and Wang (1993)

References

1. Hirayama T (1974) Prospective studies on cancer epidemiology based on census population in Japan. Proc 11th International Cancer Congress, 3, 26-35.

2. International Agency for Research on Cancer (1991) Monographs on the evaluation of carcinogenic risks to humans. Volume 51: Coffee, tea, mate, methylxanthines and methylgloyoxal, 41-197. IARC, Lyon.

3. Kinlen LJ, Willows AN, Goldblatt P et al (1988) Tea consumption and cancer. Br J Cancer, 58, 397-401.

4. Stocks P (1970) Cancer mortality in relation to national consumption of cigarettes, solid fuel, tea and coffee. Br J Cancer, 24, 215-225.

5. Tewes FJ, Koo LC, Meisgen TJ and Rylander R (1990) Lung cancer risk and mutagenicity of tea. Environ Res, 52, 23-33.

6. Yang CS and Wang Z-Y (1993) Tea and Cancer. JNCI, 85: 1038-1049.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

127. Tetrachloroethylene

Only two studies attempted to relate the risk of lung cancer to exposure to tetrachloroethylene, and details of these are given in Table 127. Both reported an excess of lung cancers but detailed results were not given for either study.

It should be noted that laundry and dry-cleaning workers may also be exposed to other solvents, especially trichloroethylene, and petroleum solvents. However, neither study gave any information on the workers' exposures. Therefore, IARC's classification of the evidence for the carcinogenicity of tetrachloroethylene to humans as "inadequate" is not really surprising [1].

Table 127: Estimates of standardized mortality ratio for exposure to tetrachloroethylene

Study	Population	Standardized mortality ratio
Blair et al (1979)[1]	Laundry/dry-cleaning workers	>100[2]
Duh and Asal (1984)[1]	US laundry/dry-cleaning workers	>100[2]

1 From International Agency for Research on Cancer (1987)

2 No details of results given

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 355-357. IARC, Lyon.

2501209092

Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

128. 2,3,7,8-Tetrachlorodibenzo-para-dioxin

Table 128 gives details of the five studies which attempted to relate the risk of lung cancer to exposure to 2,3,7,8-tetrachlorodibenzo-para- dioxin (TCDD). One study estimated a relative risk of 1.41, while another calculated a standardized mortality ratio of 111. A total of five cases of lung or bronchogenic cancer were observed in the other three studies.

Two of the studies appeared to have taken objective measurements of exposure to TCDD. In the study by Manz, TCDD concentrations in samples of precursor materials, products, waste and soil from the grounds of the plant, and in the adipose tissue of volunteers were measured, while Fingerhut analyzed serum levels of TCDD. However, these measurements only appeared to provide information on current or very recent exposure to TCDD, and historical exposure data were not available from any of the studies. It is possible, then, that inaccuracies could have been introduced due to misclassification of exposure.

While IARC classified the evidence for the carcinogenicity of TCDD to humans as "inadequate" [3], Manz et al considered the results of their study and that by Fingerhut to be supportive of the hypothesis that TCDD is a human carcinogen [4].

Table 128: Estimates of relative risk/standardized mortality ratio for exposure to 2,3,7,8-tetrachlorodibenzo-para-dioxin

Study	Population	Relative risk (95% limits)
Jirasek et al (1973,1974)[1]	Czech workers	2 bronchogenic cancers observed
Thiess and Goldman (1977)[1]	German factory workers	1 lung cancer observed
Pazderova-Vejlupkova et al (1981)[2]	Chloracne patients	2 lung cancers observed
Fingerhut et al (1991)	US chemical plant workers	111(89-137)*
Manz et al (1991)	German chemical plant workers	1.41(0.95-2.01)

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Footnote to Table 128

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1977)

2 From International Agency for Research on Cancer (1987)

References

1. Fingerhut MA, Halperin WE, Marlow DA et al (1991) Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. N Engl J Med, 324, 212-218.

2. International Agency for Research on Cancer (1977) Monographs on the evaluation of the carcinogenic risk of chemicals to man. Volume 15: Some fumigants, the herbicides 2,4-D and 2,4,5-T, chlorinated dibenzodioxins and miscellaneous industrial chemicals, 41-102. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 350-354. IARC, Lyon.

4. Manz A, Berger J, Dwyer JH et al (1991) Cancer mortality among workers in chemical plant contaminated with dioxin. The Lancet, 338, 959-964.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

129. Textile manufacturing

Table 129 gives details of the numerous studies which attempted to relate exposures in the textile manufacturing industry to the risk of lung cancer.

For general textile workers, eleven relative risks were estimated, ranging between 0.3 and 4.8, with seven of them being above 1.00. Six standardized mortality ratios (SMR) were calculated, lying in the range of 87-192, with three of these being raised. Another six studies gave proportional mortality ratios, of between 43 and 130, again with three being raised. Finally, four standardized proportional incidence ratios were given, ranging from 75-91. It can be seen that these results are somewhat equivocal, with no obvious pattern of increasing or decreasing risk being apparent.

Several of the studies gave results restricted to workers in the cotton-textile industry only. From these studies five estimates of relative risk, of 0.3-0.8, were estimated along with 15 SMRs, ranging from 27.3-117, of which only two were raised. These results are consistent with a decrease in the risk of lung cancer among respondents whose work exposes them to cotton-textiles only.

A wide variety of occupational health hazards are present in the textile industry, of which the most common is organic dust, particularly cotton dust. Other exposures include sizing agents and spinning oils used during spinning and weaving, bacteria, desiccants and defoliants found in raw cotton, pesticide residues found in raw wool, and a variety of bleaching, scouring, singeing and mercerizing agents used during fabric preparation. Chemicals are also widely used during dyeing, printing and finishing, and exposures may be highly complex [3]. It was suggested by one of the studies [7] where an increased mortality was observed that it may have been due to exposure to asbestos and mineral oils, but as little information was available from any of the studies about the nature of the subjects' exposures, it is difficult to ascertain the level of exposure, or to estimate the effects of any one substance. Also, as an increase in the risk of lung cancer was not generally seen in the evidence for textile workers it is possible that the results of the study by Zappa may have been due to chance alone.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

A number of hypotheses have been put forward to try to explain the apparent decrease in mortality in workers in the cotton-textile industry. Firstly, it has been suggested that cotton-textile workers have a lower rate of smoking than the general population, due in part to the fact that for many years such employees were not permitted to smoke at work because of an explosion hazard associated with vegetable dusts [2]. However, it has been reported that the level of smoking among cotton-textile workers is comparable to that of respondents in other occupations [2,6]. The second possible explanation is that exposure to cotton dust may stimulate stimulate mucus production, which might protect against carcinogens such as cigarette smoke [2,3,6]. A third hypothesis is based on evidence that endotoxins, whilst toxic in high concentrations, may be potent anticancer agents at the levels at which they are found in airborne cotton dust [2,3,6].

IARC [3] classify as "limited" the evidence that exposures in the textile industry as a whole increase the risk of cancer, and indeed the pattern of results reported is unimpressive. However, the possibility of a protective effect for workers in cotton-textiles appears to be much better supported.

Table 129: Estimates of relative risk/standardized mortality ratio for textile workers

Study	Population	Relative risk (95% limits)
General textile workers:		
Versluys (1949)[1]	Dutch male weavers/woollens workers	43[2]
Guralnik (1963)[1]	US male textile mill products labourers	192*
Ashley (1967)[1]	UK wool producing boroughs	87*
Menck and Henderson (1976)[1]	US male textile workers	119(p>0.05)*
Williams et al (1977)[1]	US male textile mill products workers	2.6

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 129 continued

Decoufle et al (1978)[1]	US male textile mill operatives	1.5(p>0.05)
Harrington et al (1978)[1]	US male textile workers	0.88
Newhouse (1978)[1]	UK male dyers/bleachers/textile workers	94*[3]
Buiatti et al (1979)[1]	Italian textile workers - male	2.0(1.4-2.7)
	Females	4.8(0.9-14.0)
Delzell and Grufferman (1983)[1]	US female textile workers	90[2]
Milne et al (1983)[1]	US male textile manufacturers	1.9
Coggon et al (1986)[1]	UK male textile workers	0.3
OPCS (1986)	British textile workers - male	94*
	Married women[4]	115*
Siemiatycki et al (1986)[1]	Wool workers	0.5(0.3-1.0)
	Synthetics workers	0.5(0.2-1.1)
Olsen and Jensen (1987)[1]	Danish male textile workers	88(70-111)[5]
	Females	82(56-120)[5]
	Male spinners/weavers/finishers	91(66-125)[5]
	Females	75(39-143)[5]
Paci et al (1987)[1]	Italian male nonasbestos textile workers	1.5(0.98-2.3)
Dubrow and Gute (1988)[1]	US male textile workers	<100[2]
	Service workers	130(p>0.05)[2]
	Synthetics/silk only workers	130(p>0.05)[2]
O'Brien and Decoufle (1988)[1]	US male carpet/textile workers	100(90-110)[2]
Zappa et al (1993)	Italian residents	1.45(1.0-2.1)
Cotton-textile workers:		
Kennaway and Kennaway (1936)[1]	E/W male cotton spinners/piecers	0.3
	Cotton strippers/grinders/card-room jobbers	0.3
	Cotton weavers	0.5
Enterline (1965)[6]	Male cotton-textile workers	27.3*[7]
Ashley (1967)[1]	UK cotton-textile producing towns	89*
Registrar General (1971)[8]	UK cotton-textile workers - men	85(p<0.05)*
	Single women	78*
	Married women[4]	81(p<0.05)*

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 129 continued

Henderson and Enterline (1973)[6]	Male cotton-textile workers	54.8*[7]
Daum et al (1975)	US male cotton-textile workers	93*
	Females	96*[3]
Merchant and Ortmeyer (1981)[6]	Male cotton-textile workers	40*[7]
Logan (1982)	UK male cotton spinners - 1931	117*
	1951	57*
	1961	66*
	1971	109*
	Married women[4] - 1961	91*
Siemiatycki et al (1986)[1]	Canadian cotton workers	0.8(0.4-1.3)
Levin et al (1987)[1]	Chinese cotton-textile workers	0.7(0.6-0.9)
Hodgson and Jones (1990)[8]	British cotton workers	76(54-102)*

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1990)

2 Proportional mortality ratio

3 Estimated from data given

4 According to husband's occupation

5 Standardized proportional incidence ratio

6 From Enterline et al (1985)

7 Tumours of respiratory system

8 From Rylander (1990)

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

References

1. Daum SM, Seidman H, Hammond EC and Selikoff IJ (1975) Mortality experience of a cohort of cotton textile workers. Presented at the XVIII International Congress on Occupational Health, Brighton, England, September 16th.

2. Enterline PE, Sykora JL, Keleti G and Lange JH (1985) Endotoxins, cotton dust, and cancer. The Lancet, Oct 26, 934-935.

3. International Agency for Research on Cancer (1990) Monographs on the evaluation of carcinogenic risks to humans. Volume 48: Some flame retardants and textile chemicals, and exposures in the textile manufacturing industry, 215-278. IARC, Lyon.

4. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

5. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

6. Rylander R (1990) Environmental exposures with decreased risks for lung cancer? Int J Epidemiol, 19 (Suppl 1), S67-S72.

7. Zappa M, Paci E, Seniori Constantini A and Kriebel D (1993) Lung cancer among textile workers in the Prato area of Italy. Scand J Work Environ Health, 19, 16-20.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

130. Tin miners

Three studies were found which attempted to relate lung cancer risk to employment as a tin miner, and details of these are given in Table 130. Two relative risks, of 1.01 and 2.4, were estimated and one standardized mortality ratio of 157.7 was calculated.

It was suggested that underground tin miners may be exposed to various substances, including arsenic, silica, radon and polycyclic aromatic hydrocarbons (PAHs) [1-3]. The study by Hodgson and Jones used contemporary measurements of radon to estimate historical levels, and also recorded exposure to arsenic among the subjects. McLaughlin et al measured silica concentrations as well as gathering limited information on exposure to radon, arsenic, PAHs, asbestos, nickel, talc and cadmium. The study by Wu et al measured radon levels, and dust concentrations of arsenic, iron, chromium, cadmium and nickel. However, as little information was available on lung cancer risk in relation to such exposures, and with so few studies reporting, the exact carcinogen(s) responsible for the observed increases in lung cancer risk remain unclear.

Table 130: Estimates of relative risk/standardized mortality ratio for tin miners

Study	Population	Relative risk (95% limits)
Wu et al (1989)	Chinese tin miners	2.4
Hodgson and Jones (1990)	English tin miners	157.7(p<0.01)*
McLaughlin et al (1992)	Chinese tin miners	1.01(0.77-1.33)[1]

* Standardized mortality ratio

1 Estimated from data given

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

References

1. Hodgson JT and Jones RD (1990) Mortality of a cohort of tin miners 1941-86. Br J Ind Med, 47, 665-676.

2. McLaughlin JK, Chen J-Q, Dosemeci M et al (1992) A nested case-control study of lung cancer among silica exposed workers in China. Br J Ind Med, 49, 167-171.

3. Wu K-G, Fu H, Mo C-Z, and Y L-Z (1989) Smelting, underground mining, smoking, and lung cancer: A case-control study in a tin mine area. Biomed Environ Sci, 2, 98-105.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

131. Tobacco workers

Four studies were found which gave data relevant to an investigation of a possible association between lung cancer risk and tobacco work, and details of them are presented in Table 131. Standardized mortality ratios ranging from 87-147 were calculated, with three out of five being raised. Four proportional mortality ratios were also presented, and these ranged from 112-325. Lastly, one study estimated a relative risk of 15.1.

Before interpreting these results, one potential problem in the design of the study by Blair should be noted. The subjects were selected from obituary listings of the Tobacco Workers' International Union, and a failure to include workers who dropped their Union membership before death or retirement may have biased the findings.

Tobacco workers may be exposed to various chemicals during the processing of tobacco from leaf to finished product, including residues of pesticides and barn fumigants such as phosphine gas, dichlorovinyl dimethylphosphate or carbon tetrachloride, and silica and leaf dusts from processes such as stemming, tipping and redrying [1]. However, no measurements of any of these exposures were attempted by any of the studies. Workers in the study by Talcott et al were exposed to crocidolite asbestos, but no objective measurements of the level of exposure were available.

Thus, although the evidence in the table suggests that there may be an increased risk of lung cancer among tobacco workers, no clear explanation for this observation has been put forward.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 131: Estimates of relative risk/standardized mortality ratio for tobacco workers

Study	Population	Relative risk (95% limits)
OPCS (1978)	UK male tobacco preparers and product makers, aged 15-64	87*
	Aged 65-74	142[1]
	Married women[2] aged 65-74	325[1]
	Unmarried women aged 15-64	144*
	Aged 65-74	243[1]
Logan (1982)	UK male tobacco workers - 1951	140*
	1961	147*
	1971	87*
Blair et al (1983)	US tobacco workers	112[1]
Talcott et al (1989)	US cigarette filter factory workers	15.1(7.5-27.0)[3]

* Standardized mortality ratio

1 Proportional mortality ratio

2 According to husband's occupation

3 Incidence

References

1. Blair A, Berney BW, Heid MF and White DW (1983) Causes of death among workers in the tobacco industry. Arch Environ Health, 38, 223-228.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

3. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

4. Talcott JA, Thurber WA, Arlene RN et al (1989) Asbestos-associated diseases in a cohort of cigarette-filter workers. N Engl J Med, 321, 1220-1223.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

132. Toluenes

Only three studies were found which attempted to investigate a possible association between lung cancer risk and exposure to toluenes, and details of these are given in Table 132. Two standardized mortality ratios, of 147 and 278, were calculated, while one study simply stated that six cases of respiratory cancer were observed.

Only the study by Walker made objective measurements of the concentrations of toluene the workers were exposed to, and none of the studies gave any information which would have allowed an estimation of the differential risk for individual toluenes. It is not really surprising then that IARC considered the evidence for the carcinogenicity of toluenes to humans to be "inadequate" [1].

Table 132: Estimates of relative risk/standardized mortality ratio for exposure to toluenes

Study	Population	Standardized mortality ratio
Sakabe et al (1976)[1]/ Sakabe and Fukuda (1977)[1]	Japanese workers	Six cases of respiratory cancer observed
Sorahan et al (1983)[1]	UK workers	278*[2]
Walker et al (1993)	US shoe factory workers	147(120-180)*

1 From International Agency for Research on Cancer (1987)

2 Estimated from data given

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 148-149. IARC, Lyon.

2. Walker JT, Bloom TF, Stern FB et al (1993) Mortality of workers employed in shoe manufacturing. Scand J Work Environ Health, 19, 89-95.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

133. Tuberculosis

Table 133 details the studies which attempted to relate lung cancer risk to ever having suffered from tuberculosis. Fifty relative risks were estimated, ranging from 0.84-29.82, of which 47 were above 1.00. Two standardized proportionate mortality ratios, both of 1.50, were also estimated. In addition, one study reported a positive association between lung cancer and tuberculosis but did not provide any detailed results, another observed that lung cancer occurred 20 times more frequently in those who had suffered from tuberculosis than in the general population, and one study reported that 10.0% of male and 8.2% of female lung cancer cases had a history of tuberculosis infection. Finally, one study reported finding a correlation between lung cancer mortality in adulthood and tuberculosis mortality in the period corresponding to the childhood of the lung cancer cases.

Although many of the studies gathered information on tuberculosis infection from registers or medical records, or followed-up groups of tuberculosis patients, several studies (Alavanja, Campbell, Campbell and Hughes, Wu, Wynder, Wynder and Fairchild) appeared to based their data on information elicited at interviews with the subjects. Also, it was not clear how the data on tuberculosis infection were collected in the study by Zheng. Therefore, it is possible that these studies may contain inaccuracies due to misclassification of disease status.

The data presented in the table suggest a positive association between lung cancer risk and a history of tuberculosis infection, but on the whole the estimates of relative risk are not particularly large, and therefore the relationship does not appear to be very strong. A recent review by Aoki [2] stated that although patients with active pulmonary tuberculosis had a higher than normal risk of dying from lung cancer, there was "little biological evidence of TB lesions themselves, or TB bacilli per se, having major roles to play in the carcinogenesis of lung cancer at the moment".

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 133: Estimates of relative risk for history of tuberculosis

Study	Population	Relative risk (95% limits)
Wynder et al (1956)[1]	Women	1.00
Campbell and Hughes (1960)[1]	Not stated	Lung cancer occurred 20 times more frequently in TB patients than in general population
Campbell (1961)[1]	Not stated	Positive association
Steinitz (1965)[3]	Israeli residents	10.0% male and 8.2% female lung cancer cases had history of TB
Wynder and Fairchild (1966)	US men	0.84(0.22-3.21)[2]
Simecek and Simeckova (1967)[3]	Czech residents	4.2
Aoki et al (1969)[3]	US white men	6.7(4.0-9.6)
	Non-white men	8.6(4.0-13.7)
	US white men, active TB	10.5
	Non-white men	20.6
	White men, inactive TB	4.5
	Non-white men	2.3
	US residents, 1962-3	3.6(1.9-5.3)
	1963-4	1.7(0.4-3.0)
	Japanese men	2.75
	Women	5.48
Aoki et al (1969)[3]/ Ipsen (1967)[3]	US men	3.1
Campbell and Guilfoyle (1970)[3]	Australian men	2.06
Kreus et al (1970)[3]	Finnish residents	2.2

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Table 133 continued

Aoki and Ohtani (1971)[3]	Japanese men, 1964-68, CVD controls	1.41
	Gastric cancer controls	1.23
	Women, CVD controls	1.33
	Gastric cancer controls	1.54
	Men, 1974-82, CVD controls	4.21
	IHD controls	2.13
	Women, CVD controls	3.87
	IHD controls	2.16
Watanabe and Kurashima (1977)[3]	Japanese residents	3.56
	Japanese residents	6.93
Clemmensen and Hjalgrim-Nelson (1979)[3]	Danish men, INH+	3.36
	INH-	2.58
	Women, INH+	4.55
Howe et al (1979)[3]	Canadian men	1.50(1.21-1.47)[4]
	Women	1.50(1.11-1.98)[4]
Hongo et al (1981)[3]	Japanese men	4.88
	Women	9.69
Komatsu et al (1981)[3]	Japanese men	2.58
	Women	19.6
Mercer (1981)	English residents	Correlation between lung cancer mortality in adulthood and TB mortality in time period relating to childhood
Hinds et al (1982)	Hawaiian women	1.0(0.2-5.5)
	Japanese women	2.0(0.4-10.3)
	Chinese women	2.1(0.3-16.1)
Aoki (1985)[3]	Japanese men	12.0
	Women	5.0
Takatorige et al (1985)[3]	Japanese residents, -1 year follow-up	29.82
	1-2 years	6.36
	2-3 years	5.00
	3-4 years	4.35
	4-5 years	1.27
	5+ years	1.27

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Table 133 continued

Zheng et al (1987)[3]	Chinese residents	1.5(1.2-1.8)
Wu et al (1988)	Not stated	10.0(1.1-90.1)
Sakurai et al (1989)[3]	Japanese women	6.4
Gao et al (1991)[3]	Chinese men	1.72(1.11-2.53)
	Women	2.79(1.79-4.14)
Alavanja et al (1992)	US female residents	2.0(1.0-4.1)

CVD = cardiovascular disease; IHD = ischaemic heart disease; INH = Isoniazid treatment

1 From Roe and Walters (1965)

2 Estimated from data given

3 From Aoki (1993)

4 Standardized proportionate mortality ratio

References

1. Alavanja MCR, Brownson RC, Boice JD and Hock E (1992) Preexisting lung disease and lung cancer among nonsmoking women. Am J Epidemiol, 136, 623-632.

2. Aoki K (1993) Excess incidence of lung cancer among pulmonary tuberculosis patients. Jpn J Clin Oncol, 23, 205-220.

3. Hinds MW, Cohen HI and Kolonel LN (1982) Tuberculosis and lung cancer risk in nonsmoking women. Am Rev Resp Dis, 125, 776-778.

4. Mercer AJ (1981) Risk of dying from tuberculosis or cancer: Further aspects of a possible association. Int J Epidemiol, 10, 377-380.

5. Roe FJC and Walters MA (1965) Some unsolved problems in lung cancer etiology. Progr Exp Tumor Res, 6, 126-227.

6. Wu AH, Yu MC, Thomas DC, Pike MC and Henderson BE (1988) Cancer Res, 48, 7279-7284.

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7. Wynder EL and Fairchild EP (1966) The role of a history of persistent cough in the epidemiology of lung cancer. Am Rev Resp Dis, 94, 709-720.

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134. Typhoid infection

Only one study could be found which attempted to relate the risk of lung cancer to typhoid, and details of it are given in Table 134. A relative risk of 2.5 was estimated.

This excess risk was only seen in chronic typhoid and paratyphoid carriers, and not in a group of people who had suffered from the acute disease but not gone on to become carriers. It was therefore suggested by the authors that an increased lung cancer risk is only associated with chronic carriers, but with only one study reporting there is no other evidence to support this view, or indeed that typhoid in any form is a risk factor for lung cancer.

Table 134: Estimates of relative risk for typhoid infection

Study	Population	Relative risk (95% limits)
Caygill et al (1994)	Scottish residents	2.5(0.82-5.89)

References

1. Caygill CPJ, Hill MJ, Braddick M and Sharp JCM (1994) Cancer mortality in chronic typhoid and paratyphoid carriers. Lancet, 343, 83-84.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

135. Uranium compounds

Table 135 gives details of the studies found which provided data relevant to an investigation of the possible association between lung cancer risk and exposure to uranium compounds. Fifteen standardized mortality ratios were calculated, ranging from 82-482, with 12 being raised. One study estimated relative risks of between 0.943 and 1.027 per centigray increase in the cumulative lung dose of uranium, while two others stated that excesses of lung cancer had been observed, but failed to give any detailed results.

Various drawbacks were noted in some of the studies, which should be highlighted before an evaluation of the evidence is made. The standardized mortality ratio presented by Archer was calculated on the basis of nonwhite rates of lung cancer, and it was stated that this would have produced an overestimate of the expected number of lung cancers in the study population. It was also noted that there was a degree of overlap in the studies by Chovil, Ham and Muller, which cannot therefore be considered as completely independent of each other. Other potential problems include possible incomplete ascertainment of cases in the studies by Chovil and Kunz.

It was noted in the study by Kusiak that many of the miners were also exposed to arsenic, nickel, copper and gold, and that there was a difference in risk particularly for those miners who had also been exposed to arsenic and gold. Additionally, it is highly likely that most of the subjects in these studies were exposed to radiation, and it is possible that this is a more important factor in the observed increase in in lung cancer than the uranium itself. In fact, the National Research Council stated that "the dominant source of radiation damage to the respiratory system in miners is generally considered to be the inhalation of radon daughters, rather than the uranium content of the ore". Therefore, it is difficult to properly determine the carcinogenicity of uranium and its compounds to humans, although the evidence presented in the table is suggestive of a positive association.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 135: Estimates of standardized mortality ratio/relative risk for uranium compounds

Study	Population	Standardized mortality ratio (95% limits)
Archer et al (1976)[1]	American Indian uranium miners	423(p>0.01)
Ham (1976)[1]	Canadian uranium miners	180[2]
Kunz et al (1979)[1]	Czech uranium miners	Excess lung cancer observed for all levels of cumulative exposure and exposure duration
Chovil (1981)[1]	Canadian uranium miners	Excess of lung cancer observed
Muller et al (1981, 1983,1985)[1]	Canadian uranium miners	181(p<0.05)
Waxweiler et al (1981)[1]	US white uranium miners	482(lower 95% limit 425)
Waxweiler et al (1983)[3]	US uranium workers never employed in mining	83(54-121)
Tirmarche et al (1984)[1]	French uranium miners	191[2]
Nair et al (1985)[3]	Canadian uranium miners	184
	Uranium refinery workers	82
	Surface uranium miners	175
	Underground uranium miners	375
	Other uranium workers	110
Howe et al (1986)[1]	Canadian uranium miners	190[2]
Polednak and Frome (1986)[3]	US uranium processing workers employed less than 1 year	92
	Employed 1 year or more	106
Dupree et al (1993)	US uranium processing workers	0.943-1.027[4]
Kusiak et al (1993)	Canadian uranium miners	225(191-264)

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Footnote to Table 135

1 From International Agency for Research on Cancer (1988)

2 Estimated from data given

3 From National Research Council (1988)

4 Relative risk per centigray increase in cumulative lung dose

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References

1. Dupree E, Watkins J, Ingle J et al (1993) Risk of lung cancer among uranium processing workers. Am J Epidemiol, 138, 640 (Abstract).

2. International Agency for Research on Cancer (1988) Monographs on the evaluation of carcinogenic risks to humans. Volume 43: Man-made mineral fibres and radon, 173-259. IARC, Lyon.

3. Kusiak RA, Ritchie AC, Muller J and Springer J (1993) Mortality from lung cancer in Ontario uranium miners. Br J Ind Med, 50, 920-928.

4. National Research Council (1988) Health risks of radon and other internally deposited alpha-emitters: BEIR IV. National Academy Press, Washington DC.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

136. Vegetarianism

Table 136 gives details of the two studies which investigated lung cancer risk in relation to a vegetarian diet. Three standardized mortality ratios, ranging from 8-89, were calculated.

While these results are indicative of a protective effect of vegetarianism, data from more studies is needed before such a relationship can be confirmed.

Table 136: Estimates of standardized mortality ratio for vegetarianism

Study	Population	Standardized mortality ratio
Kinlen (1982)	UK female members of religious orders	45
Chang-Claude and Frentzel-Beyme (1993)	German men	8
	Women	89

References

1. Chang-Claude J and Frentzel-Beyme R (1993) Dietary and lifestyle determinants of mortality among German vegetarians. Int J Epidemiol, 22, 228-236.

2. Kinlen LJ (1982) Meat and fat consumption and cancer mortality: A study of strict religious orders in Britain. Lancet, 1, 946-949.

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

137. Vinyl chloride

Although most of the studies which have investigated the potential effects of exposure to vinyl chloride have concentrated on liver cancer, a few studies were found which had also looked for a possible association with lung cancer, and details of these are given in Table 137. Few detailed results were available, but it can be seen from the table that of the 10 estimates of relative risk given, all but 1 were raised. Additionally, one standardized mortality ratio, of 194, was presented.

It is not clear how extensive the exposures of the subjects were, as objective measurements of vinyl chloride concentrations in the workplace do not appear to have been made by any of the studies. It is also possible that some workers may have been exposed to other chemicals which may themselves be potential carcinogens. Lastly, there are differences between the studies in the length of time a subject had to be employed for before being considered as "exposed", which may also introduce inaccuracies.

Additionally, the findings by Monson can be questioned, on the grounds that not all of the deaths studied had occurred in workers in activities directly related to vinyl chloride production or polymerization and that the study failed to include deaths among workers who had terminated employment prior to retirement or death [1].

Despite these drawbacks, IARC considered there to be "sufficient" evidence for the carcinogenicity of vinyl chloride to humans [1,2].

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Source: https://www.industrydocuments.ucsf.edu/docs/yhhl0000

Table 137: Estimates of relative risk/standardized mortality ratio for exposure to vinyl chloride

Study	Population	Relative risk (95% limits)
Monson et al (1974)[1]	US vinyl chloride workers	>1.00
Tabershaw and Gaffey (1974)[1]	US vinyl chloride workers	>1.00[2]
Byren et al (1976)[1]	Swedish vinyl chloride/polyvinyl chloride workers	>1.00(p>0.05)
Fox and Collier (1976)[1]	Workers exposed to vinyl chloride for <15 years	1.56
Saric et al (1976)[1]	Yugoslavian vinyl/polyvinyl chloride workers	1.00
Waxweiller et al (1976)[1]	US vinyl chloride/polyvinyl chloride workers	194*[2]
Von Reinl et al (1977)[1]	German vinyl chloride workers	>1.00
Buffler et al (1979)[3]	Vinyl chloride workers	>1.00
Waxweiler et al (1981)[3]	Synthetic chemical plant workers	>1.00
Fedotova (1983)[3]	Vinyl chloride/polyvinyl chloride workers	>1.00
Heldaas et al (1984)[3]	Vinyl chloride/polyvinyl chloride workers	>1.00

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1979)

2 Tumours of respiratory system

3 From International Agency for Research on Cancer (1987)

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References

1. International Agency for Research on Cancer (1979) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 19: Some monomers, plastics and synthetic elastomers, and acrolein, 377-438. IARC, Lyon.

2. International Agency for Research on Cancer (1979) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 373-376. IARC, Lyon.

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138. Vitamin A

There has recently been much interest in the relationship between diet and the risk of cancer, and several studies have attempted to investigate the possible association between vitamin A intake and the risk of lung cancer. The results of these studies are detailed in Table 138, which also includes two overall estimates of relative risk, based on 3 and 24 studies respectively. For those studies which divided the respondents into multiple categories of vitamin A intake only the relative risk estimate for the highest, or lowest, intake category has been given, as appropriate. For the studies which used a high intake of vitamin A as the reference category, 11 out of 13 estimates of relative risk were above 1.00, ranging from 1.13-3.00. The other two studies failed to find an association, but did not report their results in detail. All of the 7 relative risks presented from the studies which compared a high intake of vitamin A to a base category of low intake were reduced, lying in the range of 0.31-0.91.

It can be seen from the table that the index of vitamin A used varied widely. A number of studies took measurements from blood samples, recording serum levels of vitamin A or beta-carotene. Most of the remaining studies concentrated on dietary intake, calculating indices of vitamin A or beta-carotene intake from the amount of certain foods eaten, although three studies simply used total consumption of vegetables, or fruits and vegetables, as an index of vitamin A intake. It is not clear how reliable indices of vitamin A intake based on dietary assessment are, especially where the frequency of consumption rather than the actual amount eaten is measured. Additionally, there may be important sources of vitamin A, such as vitamin supplements, which are not included in dietary questionnaires.

Another point to consider is the fact that vitamin A is taken into the body in two forms; as pre-formed vitamin A and as beta-carotene, from which the body is able to manufacture the vitamin as required. Therefore, it is difficult to tell whether it is the vitamin itself or the provitamin which is providing the protective effect. One further problem lies in the fact that foods are rarely a source for just one vitamin but

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usually contain several. A high intake of vitamin A, therefore, could correlate with a high intake of some other component of the diet which may itself be the protective factor, rather than the vitamin.

Finally, the development of cancer may affect appetite and change the way the body metabolizes food. As a result, cancer patients may appear to have different levels of serum and dietary vitamins than those not suffering from cancer, but these could be as a result of the disease rather than a cause of it. Therefore, case-control studies of the relationship between diet and cancer should be viewed with some caution, while cohort studies, which measure vitamin levels or dietary intake before the onset of disease, may provide more reliable results.

Bearing all these potential problems in mind, the results obtained from the various studies are remarkably consistent, suggesting that there may indeed be some protective effect, although whether this is from vitamin A or beta-carotene is not clear.

Table 138: Estimates of relative risk for vitamin A intake

Study	Study type	Location	Vitamin A index	Relative risk (95% limits)
Highest intake as reference category:				
MacLennan et al (1977)	C-C	Singapore	VEG	2.23(1.49-3.33)
Hankin et al (1984)	C-C	USA	DVA	1.39(0.89-2.15)
Seigel (1984)	C-C[1]	USA/Switzerland	SVA	No association
Menkes et al (1986)	PR	USA	SVA	1.13
			SBC	2.20
Humble et al (1987)	C-C	USA	DVA	1.29(0.91-1.83)
			DBC	1.35(0.96-1.90)
Kok et al (1987)	PR	Netherlands	SVA	No association
Orentreich et al (1991)	C-C	USA	SVA	1.5
			SBC	3.0
Block et al (1992)	CO[2]	Various	FVI	2.20(1.20-7.00)
Forman et al (1992)	C-C	China	FVI	1.22(0.76-1.94)
Dorgan et al (1993)	C-C	USA	DCA	1.27(1.03-1.56)

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Table 138 continued

Lowest intake as reference category:

Bjelke (1975)	PR	Norway	DVA	0.31 ($p < 0.01$)
Wald et al (1988)	PR	England	SBC	0.81(0.37-1.78)
Chow et al (1992)	PR	USA	DVA	0.80(0.50-1.20)
			DBC	0.80(0.50-1.20)
Huang et al (1992)	C-C	China	DBC	0.91(0.85-0.99)
Shibata et al (1992)	PR	USA	DBC	0.81(0.49-1.33)
Steinmetz et al (1993)	PR	USA	DBC	0.83(0.52-1.32)

C-C = Case-control; CO = Combined; DBC = Dietary beta-carotene; DCA = Dietary carotenoids; DVA= Dietary vitamin A; FVI = Fruit and vegetable intake; PR = Prospective; SBC = Serum beta-carotene; SVA = serum vitamin A; VEG = Vegetable intake;

1 Combined results from 3 studies

2 Combined results from 24 studies

References

1. Bjelke E (1975) Dietary vitamin A and human lung cancer. Int J Cancer, 15, 561-565.

2. Block G, Patterson B and Subar A (1992) Fruit, vegetables and cancer prevention: a review of the epidemiological evidence. Nutr Cancer, 18, 1-29.

3. Dorgan JF, Ziegler RG, Schoenberg JB et al (1993) Race and sex differences in associations of vegetables, fruits, and carotenoids with lung cancer risk in New Jersey (United States). Cancer Causes and Control, 4, 273-281.

4. Forman MR, Yao SX, Graubard BI et al (1992) The effect of dietary intake of fruits and vegetables on the odds ratio of lung cancer among Yunnan tin miners. Int J Epidemiol, 21, 437-441.

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139. Vitamin C

Details of some 14 studies which have attempted to investigate the possible association between vitamin C intake and the risk of lung cancer are given in Table 139, along with the key findings. Where the respondents were divided into multiple categories of vitamin C intake only the relative risk estimate for the highest, or lowest, category has been given, as appropriate. Thirteen out of the 17 estimates of relative risk presented from studies which used a high intake of vitamin C as the reference category showed a positive association, with risks ranging from 1.28-4.30, although several studies did not give details of their results. The remaining four studies failed to find an association. All of the 4 relative risks presented from the studies which compared a high intake of vitamin C to a base category of low intake were reduced, lying in the range of 0.26-0.94.

The table shows the indices of vitamin C intake used by the studies, and it can be seen that these varied widely. Only one study took measurements of serum vitamin C levels. The others measured dietary intake, calculating indices of vitamin C intake from the amount of certain foods eaten, although three studies also looked at total consumption of fruit or green salads. The reliability of nutrient indices based on dietary assessment is unclear, especially where only the frequency of consumption is measured, rather than the actual amount eaten. Additionally, as only three studies recorded usage of vitamin supplements intake of vitamin C from this source will probably not have been included by many of the studies.

One further problem lies in the fact that foods are rarely a source for just one vitamin but usually contain several. A high intake of vitamin C, therefore, could correlate with a high intake of some other component of the diet which may itself be the protective factor. In particular, foods high in vitamin C are also often high in carotenoids, for which there is some evidence of a protective effect [1]. Alternatively, a high intake of foods containing vitamin C may correlate with a low intake of foods containing nutrients, such as fat, which are believed to be positively associated with the risk of cancer.

Finally, due to the fact that the development of cancer might affect appetite and change the way the body metabolizes food, cancer patients

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5. Hankin JH, Kolonel LN and Hinds MW (1984) Dietary history methods for epidemiologic studies: application in a case-control study of vitamin A and lung cancer. JNCI, 73, 1417-1421.

6. Huang C, Zhang X, Qiao Z et al (1992) A case-control study of dietary factors in patients with lung cancer. Biomed Environ Sci, 5, 257-265.

7. Humble CG, Samet JM and Skipper BE (1987) Use of quantified and frequency indices of vitamin A intake in a case-control study of lung cancer. Int J Epidemiol, 16, 341-346.

8. Kok FJ, van Duijn CM, Hofman A, Vermeeren R, de Bruijn AM and Valkenburg HA (1987) Micronutrients and the risk of lung cancer (Letter). N Engl J Med, 316, 1416.

9. MacLennan R, Da Costa J, Day NE, Law CH, Ng YK and Shanmugaratnam K (1977) Risk factors for lung cancer in Singapore Chinese, a population with high female incidence rates. Int J Cancer, 20, 854-860.

10. Menkes MS, Comstock GW, Vuilleumier JP, Helsing KJ, Rider AA and Brookmeyer R (1986) Serum beta-carotene, vitamins A and E, selenium, and the risk of lung cancer. New Engl J Med, 315, 1250-1254.

11. Orentreich N, Matias JR, Vogelman JH, Salkeld RM, Bhagavan H and Friedman GD (1991) The predictive value of serum β-carotene for subsequent development of lung cancer. Nutr Cancer, 16, 167-169.

12. Seigel D (1984) Discussion of case-control studies of Peleg, Stahelin, and Willett. JNCI, 73, 1469-1470.

13. Shibata A, Paganini-Hill A, Ross RK, Yu MC and Henderson BE (1992) Dietary β-carotene, cigarette smoking, and lung cancer in men. Cancer Causes and Control, 3, 207-214.

14. Steinmetz KA, Potter JD and Folsom AR (1993) Vegetables, fruit, and lung cancer in the Iowa Women's Health Study. Cancer Res, 53, 536-543.

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may appear to have different levels of serum and dietary vitamins than those not suffering from cancer, as a result of the disease rather than as a cause of it. For this reason, cohort studies which measure vitamin levels or dietary intake before the onset of disease may provide more reliable results than studies of a case-control design.

Despite these problems the results obtained are fairly consistent over the various studies, suggesting that vitamin C may indeed have a protective effect. However, due to the failure of several of the studies to present their results in detail, it is difficult to estimate the strength of such an effect if it does exist.

Table 139: Estimates of relative risk for vitamin C intake

Study	Study type	Location	Vitamin C index	Relative risk (95% limits)
Low vitamin C levels:				
Mettlin et al (1979)	C-C	USA	DVC	1.00
Shekelle et al (1981)*	PR	Not given	DVC	>1.00 (p >0.05)
Kvale et al (1983)	PR	Norway	DVC	1.00
Hinds et al (1984)	C-C	USA	DIS	1.60 (p >0.05)[1]
Kolonel et al (1985)	C-C	USA	DIS	1.28(0.69-2.38)[2]
Byers et al (1987)	C-C	USA	DVC	1.00
Kromhout (1987)*	PR	Not given	DVC	2.80 (p <0.01)
			FRU	>1.00 (p <0.05)
Stahelin et al (1987)*	PR	Not given	SVC	>1.00 (p >0.05)
Fontham et al (1988)	C-C	USA	DVC	1.50(p <0.001)
Holst et al (1988)	C-C	Netherlands	DVC	4.30
Koo (1988)	C-C	Hong Kong	FRU	2.19(1.22-5.55)
			DVC	1.96(1.15-4.76)
Le Marchand et al (1989)	C-C	USA	DIS	>1.00[1]
			DVC	>1.00[1]
			DIS	1.00[3]
			DVC	>1.00 (p >0.05)[3]

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Table 139 continued

High vitamin C levels:				
Fraser et al (1991)	PR	USA	FRU	0.26(0.10-0.70)
			SAL	0.65(0.29-1.47)
Enstrom et al (1992)	PR	USA	DIS	0.94(0.52-1.71)[2]
			DVC	0.73(0.51-1.04)[2]

* Taken from Block (1991)

1 Males only

2 Estimated from data for males and females separately

3 Females only

C-C = Case-control; DIS = Dietary vitamin C, including supplements; DVC = Dietary vitamin C; FRU = Fruit intake; PR = Prospective; SAL = Green salads; SVC = Serum vitamin C

References

1. Block G (1991) Vitamin C and cancer prevention: the epidemiologic evidence. Am J Clin Nutr, 53, 270S-282S.

2. Byers T, Vena J, Mettlin C, Swanson M and Graham S (1984) Dietary vitamin A and lung cancer risk: an analysis by histological subtypes. Am J Epidemiol, 120, 769-776.

3. Enstrom JE, Kanim LE and Klein MA (1992) Vitamin C intake and mortality among a sample of the United States population. Epidemiol, 3, 194-202.

4. Fontham ETH, Pickle LW, Haenszel W, Correa P, Line Y and Falk RT (1988) Dietary vitamins A and C and lung cancer risk in Louisiana. Cancer, 62, 2267-2273.

5. Fraser GE, Beeson WL and Phillips RL (1991) Diet and lung cancer in California Seventh-Day Adventists. Am J Epidemiol, 133, 683-693.

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6. Hinds MW, Kolonel LN, Hankin JH and Lee J (1984) Dietary vitamin A, carotene, vitamin C and risk of lung cancer in Hawaii. Am J Epidemiol, 119, 227-237. (with 1)

7. Holst PA, Kromhout D and Brand R (1988) For debate: pet birds as an independent risk factor for lung cancer. Br Med J, 297, 1319-1321.

8. Kolonel LN, Hinds MW, Nomura AMY, Hankin JH and Lee J (1985) Relationship of dietary vitamin A and ascorbic acid intake to the risk for cancers of the lung, bladder, and prostate in Hawaii. Natl Cancer Inst Monogr, 69, 137-142.

9. Koo LC (1988) Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoked. Nutr Cancer, 11, 155-172.

10. Kromhout D (1987) Essential micronutrients in relation to carcinogenesis. Am J Clin Nutr, 45, 1361-1367.

11. Kvale G, Bjelke E and Gart JJ (1983) Dietary habits and lung cancer risk. Int J Cancer, 31, 397-405.

12. Le Marchand L, Yoshizawa CN, Kolonel LN, Hankin JH and Goodman MT (1989) Vegetable consumption and lung cancer risk: a population-based case-control study in Hawaii. JNCI, 81, 1158-1164.

13. Mettlin C, Graham S and Swanson M (1979) Vitamin A and lung cancer. JNCI, 62, 1435-1438.

14. Shekelle RB, Lepper M, Liu S et al (1981) Dietary vitamin A and risk of cancer in the Western Electric study. Lancet, 2, 1185-1190.

15. Stahelin HB, Gey KF and Brubacher G (1987) Plasma vitamin C and cancer death: the Prospective Basel Study. Ann NY Acad Sci, 498, 124-131.

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140. Vitamin E

Details of three studies, reporting on ten populations, which attempted to relate lung cancer risk to vitamin E levels are given in Table 140. A relative risk of 1.47 was estimated by one of the studies. In addition, in five of the study groups serum vitamin E levels were lower in cases than controls, while in two groups the reverse was true. No association was reported for the other two populations.

It has been suggested that vitamin E may inhibit the formation of nitrosamines, and therefore carcinogenesis [3], but data from more studies is needed before a protective effect can be confirmed.

Table 140: Estimates of relative risk for high vitamin E intake

Study	Population	Relative risk
Fontham (1990)	Not stated	Serum vitamin E lower in cases than controls ($p<0.05$)
	Not stated	No association reported
	Hawaiians of Japanese ancestry	No association reported
Comstock et al (1992)	US residents	Serum vitamin E lower in cases than controls ($p<0.05$)
	Swiss residents	Serum vitamin E lower in cases than controls ($p<0.05$)
	US residents	Serum vitamin E lower in cases than controls ($p>0.05$)
	Finnish men	Serum vitamin E lower in cases than controls ($p>0.05$)
	Finnish women	Serum vitamin E higher in cases than controls ($p>0.05$)
	Hawaiian residents	Serum vitamin E higher in cases than controls ($p>0.05$)
Alavanja et al (1993)	US white women	1.47

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References

1. Alavanja MCR, Brown CC, Swanson C and Brownson RC (1993) Saturated fat intake and lung cancer risk among nonsmoking women in Missouri. JNCI, 85, 1906-1916.

2. Comstock GW, Bush TL and Helzlsouer K (1992) Serum retinol, beta-carotene, vitamin E, and selenium as related to subsequent cancer of specific sites. Am J Epidemiol, 135, 115-121.

3. Fontham ETH (1990) Protective dietary factors and lung cancer. Int J Epidemiol, 19 (Suppl 1), S32-S42.

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141. Waiters

Details of the two studies found which looked for a possible association between lung cancer risk and employment as a waiter/waitress are given in Table 141. One study gave a standardized mortality ratio of 156, while the other presented a standardized incidence ratio of 2.0.

It was suggested that the increase in lung cancer among waiters may be explained by their lifestyle, particularly their smoking and drinking habits [1]. However, with so few studies reporting it is difficult to reach any firm conclusions.

Table 141: Estimates of standardized mortality/incidence ratio for working as a waiter/waitress

Study	Population	Standardized mortality ratio
Logan (1982)	UK unmarried female waitresses, 1961	156
Kjaerheim and Andersen (1993)	Norwegian male waiters	2.0(1.3-2.9)[1]

1 Standardized incidence ratio

References

1. Kjaerheim K and Andersen A (1993) Incidence of cancer among male waiters and cooks: Two Norwegian cohorts. Cancer Causes and Control, 4, 419-426.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

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142. Welding fumes

Table 142 gives details of the studies which investigated a possible association between lung cancer risk and exposure to welding fumes. For studies based on mortality/morbidity statistics, eight standardized mortality ratios (SMR) were given ranging from 92-151, of which seven were raised. Three proportional mortality ratios (PMR), of 100-145, were also presented, with two of these being raised. Three studies estimated relative risks (RR) which ranged from 0.85-3.5, of which two were above 1.00. Additionally, one study calculated an incidence rate of 125.8 per 100 000 per year, compared to the general male population. Eight SMRs were calculated from cohort studies and they ranged from 95-249, with seven being raised. The four RRs estimated lay in the range 1.25-4.4, while the two calculations of standardized incidence ratio were of 115 and 142. One study calculated a PMR of 104. Finally, 13 estimates of RR were made by case-control studies, and these ranged from 0.7-13.2, of which 11 were above 1.00.

Before an evaluation of the carcinogenicity of exposure to welding fumes is made, several potential drawbacks in the design of some of the studies should be pointed out. Firstly, the case-control studies suffer from the problem that although many of them investigated a long list of occupations and exposures most only reported those with which positive associations were found. Thus, the possibility of a publication bias in in favour of positive associations must be taken into account when reviewing these studies. Problems were also noted with some of the cohort studies. The study by Dunn et al/Dunn and Weir appeared to have incompletely followed up its subjects, while selection bias cannot be ruled out of the study by Becker et al, due to the inclusion of only those subjects who had undergone a technical examination.

Welding can be performed under a wide variety of industrial settings and therefore welders are potentially exposed to a great number of substances, derived from the welding process itself or from other industrial activities being performed in the immediate vicinity. Compounds which have been reported in welding fumes include chromium, nickel, fluoride, lead, aluminium, barium, and various gases and organic constituents [2]. However, as little information is available from the studies about the subjects' exposures it is not possible to separate out

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the effects of any one compound. Furthermore, it was suggested that subjects in several of the studies (Becker, Dunn, McMillan and Pethybridge, Puntoni) may have been exposed to asbestos from sources other than welding, which could have biased the results [2].

Taking all this into account, it is not entirely surprising that IARC classified the evidence for the carcinogenicity of welding fumes as "limited" [2].

Table 142: Estimates of relative risk/standardized mortality ratio for exposure to welding fumes

Study	Population	Relative risk (95% limits)
Mortality/morbidity statistics:		
Guralnick (1963)[1]	US welders/flame cutters	92(64-129)*
Menck and Henderson (1976)[1]	US welders	137(101-182)*
Milham (1976)[1]	US welders/flame cutters	104(92-118)[2]
Decoufle et al (1977)[1]	US welders/flame cutters	0.85
Gottlieb (1980)[1]	US welders	3.5
Petersen and Milham (1980)[1]	US welders/flame cutters	100[2]
Logan (1982)	UK male welders - 1951	118*
	1961	122*
	1971	151*
	Married women[3] - 1961	132*
	1971	107*
Morton and Treyve (1982)[1]	US welders/burners	125.8[4]
Milne et al (1983)[1]	US welders	1.2
Gallagher and Threlfall (1983)[1]	Canadian welders	145(115-183)[2]
OPCS (1986)[1]	UK male welders	146*
Cohort studies:		
Dunn et al (1960)[1]/Dunn and Weir (1965,1968)[1]	US welders/burners	105(78-139)*
Puntoni et al (1979)[1]	Italian oxyacetylene welders	1.25(0.34-3.20)
	Electric arc welders	1.60(0.33-4.66)

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Table 142 continued

Beaumont and Weiss (1980, 1981)[1]	US welders	132(98-174)*
Sjogren (1980)[5]	Swedish stainless steel welders	4.4
Polednak (1981)[1]	US welders	150(87-240)*
Fletcher and Ades (1984)[1]	UK welders	146(62-288)*
McMillan and Pethybridge (1984)[1]	UK welders	104(34-243)[2,6]
Becker et al (1985)[1]	German stainless steel welders	95(35-207)*
Newhouse et al (1985)[1]	UK welders	191*
Sjogren and Carstensen (1986)[1]	Swedish welders/gas cutters	1.3
Sjogren et al (1987)[1]	Swedish stainless-steel welders	249(80-581)*
Tola et al (1988)[1]	Finnish shipyard welders	115(76-167)[7]
	Machine shop welders	142(77-237)[7]
IARC (1989)[1]	European welders	134(110-160)*
Case-control studies:		
Breslow et al (1954)[1]	US welders/sheet metal workers doing welding	7.2(1.9-44.3)
Blot et al (1978)[1]	US welders/burners	0.7
Blot et al (1980)[1]	US welders/burners	0.9(0.4-2.3)
Buiatti et al (1985)[1]	Italian male welders	2.8(0.9-8.5)
Silverstein et al (1985)[1]	US welders/millwrights	13.2(1.1-154.9)
Gerin et al (1986)[1]	Canadian welders	2.4(1.0-5.4)
Kjuus et al (1986)[1]	Norwegian welders	1.9(0.9-3.7)
Lerchen et al (1987)[1]	US welders	3.2(1.4-7.4)
Schoenberg et al (1987)[1]	US welders/flame cutters	1.2(0.8-1.9)
Benhamou et al (1988)[1]	French welders/flame cutters	1.4(0.79-2.9)
Rinsky et al (1988)[1]	US shipyard workers with probable exposure to welding fumes	1.1(0.8-1.7)
Ronco et al (1988)[1]	Italian welders	2.9(0.87-9.8)
Keller and Howe (1993)	US construction workers	1.68(1.03-2.76)

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Footnote to Table 142

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1990)

2 Proportional mortality ratio

3 According to husband's occupation

4 Incidence per 100 000 per year

5 From International Agency for Research on Cancer (1987)

6 Tumours of respiratory system

7 Standardized incidence ratio

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 165-168. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Volume 49: Chromium, nickel and welding, 447-525. IARC, Lyon.

3. Keller JE and Howe HL (1993) Cancer in Illinois construction workers: A study. Am J Ind Med, 24, 223-230.

4. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

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143. Wood industries

Although several studies have reported an increased risk of nasal cancer in workers in wood industries, the data concerning a possible association with lung cancer is somewhat more sparse. Details of those studies which have provided information are given in Table 143.

For workers in unspecified occupations within the wood industry two relative risks, of 1.0 and 3.3, were estimated, along with three standardized mortality ratios (SMR) ranging from 104-113, and a proportional mortality ratio (PMR) of 87.

Results were also presented according to the specific occupations of the subjects. The relative risks estimated for lumber/sawmill workers appeared to show some evidence of an increase in risk, ranging from <1.0-1.7, with two of them being above 1.00. However, the two SMRs calculated, of 33 and <80, did not.

For furniture/cabinet makers two standardized mortality ratios were given, of 96 and 110. In addition two relative risks, of 0.87 and 6.0, were presented. With so few studies reporting, it is difficult to see a clear pattern of increased or decreased risk.

Four SMRs were calculated for carpenters, ranging from 96-120, with three being raised. In addition, two PMRs, of 104 and 107 were given, along with two proportional registration ratios, of 111 and 115, and one relative risk of 0.87. These results provide limited evidence of a small increase in the risk of lung cancer among carpenters/joiners.

Lastly, the six relative risks estimated for pulp/paper workers lay in the range 1.0-2.2, with four being raised. Three SMRs were given, of between 69 and 91, along with one PMR of 100. Additionally, one study stated that the lung cancer rate was 9% higher among pulp/paper workers, although no details of the comparison population were given. It can be seen that no clear pattern of increased or decreased risk emerged.

Employees in the wood industry may be exposed to various substances, depending on the nature of their occupation and the processes it involves, which may vary from country to country. The most important exposures are wood dust, solvents and biological factors such as insects and fungal spores in untreated wood. Exhaust gases and oil mists associated with sawblade lubrication are an additional hazard for workers in sawmills, while pulp/paper workers may be exposed to numerous chemicals including gaseous sulphur compounds, chlorine and chlorine dioxide, turpentine, sodium hydroxide mist, methanol, ethanol, sulphuric

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acid, furfural, hydroxymethylfurfural, cymene, acetic acid, formic acid, gluconic acid, aldonic acid and hydrogen peroxide, dusts consisting of lime, sodium sulphate, and compounds used for the control of slime and algae [1,7]. However, little information was available from the studies on the subjects' exposures, and in some of the studies there was a possibility that workers may have been employed in more than one area of the wood industry. Additionally, exposures in the various occupations within the industry are quite different, and therefore it may not be appropriate to classify workers from different occupations together, as some of the studies did.

Although IARC has classified the evidence for the association between nasal cancer and employment in furniture/cabinet-making as "sufficient", it was felt that there was not enough data available to make an evaluation of the possible association between lung cancer and employment in specific occupations within the wood industry [1].

Table 143: Estimates of relative risk/standardized mortality ratio for employment in the wood industry

Study	Population	Relative risk(95% limits)
General woodworkers:		
Harrington et al (1978)[1]	US wood/paper workers - urban	1.00
	Rural	3.3
OPCS (1978)	English/Welsh male woodworkers	113*
OPCS (1986)	UK male woodworkers, pattern makers	104*
	Married women[2]	105*
Rylander (1990)	US male mill/furniture/match/shingle/ weaver workers	87[3]
Lumber/sawmill workers:		
Milham (1974)[1]	US lumber/sawmill workers/millmen	<80*
Blot and Fraumeni (1976)[1]	US counties with lumber industries	<1.00
Harrington et al (1978)[1]	US sawmill/lumber/forestry workers	>1.00
Edling and Granstam (1980)[4]	Swedish lumberjacks	33*
Blot et al (1982)[5]	US lumber/wood workers	1.7

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Table 143 continued

Furniture/cabinet makers:		
Brinton et al (1976)[1]	US furniture/fixture makers	0.87
Olsen and Sabroe (1979)[4]	Danish male carpenters/cabinet makers, aged 20-64	96(68-114)*
	Aged 65-84	110(92-127)*
Esping and Axelson (1980)[1]	Swedish furniture makers	6.0
Carpenters/joiners:		
Milham (1974)[1]	US carpenters/joiners	106.7*
Decoufle et al (1977)[1]/ Bross et al (1978)[1]	US carpenters	0.87
Harrington et al (1978)[1]	US carpenters	>1.00
OPCS (1978)	E/W male carpenters/joiners, aged 15-64	107[3]
	Aged 65-74	104[3]
	Incidence 1966-7	111($p<0.05$)[6]
	1968-9	115($p<0.01$)[6]
Olson and Sabroe (1979)[4]	Danish male carpenters/cabinet makers, aged 20-64	96(68-114)*
	Aged 65-84	110(92-127)*
Stellman and Garfinkel (1984)[4]	Male carpenters/joiners	120($p<0.05$)*
Paper workers:		
Blot and Fraumeni (1976)[1]	US southern/eastern counties with pulp/paper industries - males	Lung cancer rate 9% higher ($p<0.05$)
Menck and Henderson (1976)[1]	US paper manufacturing/sales workers	1.71($p<0.01$)
Blot et al (1978)[1]	US paper mill workers	1.0
Gottlieb et al (1979)[1]	US paper industry workers	1.05(0.79-1.40)
Logan (1982)	UK male paper makers - 1951	69*
	1961	84*
	1971	91*
Milham and Demers (1974)[4]	US pulp/paper workers	100[3]
Nurminen and Hernberg (1984)[4]	Pulp/paper workers	2.2(0.7-6.7)

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Table 143 continued

Robinson et al (1986)[4]	US pulp/paper workers	1.00
Toren et al (1991)	Swedish paper mill workers	1.1(0.3-1.3)

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* Standardized mortality ratio

1 From International Agency for Research on Cancer (1981)

2 According to husband's occupation

3 Proportional mortality ratio

4 From International Agency for Research on Cancer (1987)

5 From Williams Pickle (1984)

6 Proportional registration ratio

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